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Original article

Psittacosis contagion in 1930: an old story in a new era of zoonotic disease

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ABSTRACT

The SARS-CoV-2 pandemic has highlighted the importance of zoonotic diseases. Psittacosis, a human disease resulting from infection spill-over from *Chlamydia psittaci*-infected birds, is a lesser-known example of a zoonosis. Psittacosis was responsible for numerous outbreaks in the 1930s, characterised by significant human mortality and disruption to the global trade in parrots. This paper describes the epidemiological and clinical details of one family group impacted by the purchase of an infected, imported parrot. Findings are discussed in the context of a growing awareness of the health risks of global disease outbreaks, as well as social and economic impacts.

Health information recorded for cases of psittacosis associated with the 1930 cluster was reviewed using contemporary knowledge of disease symptoms and epidemiology. Case details and autopsy reports were examined.

Public health investigation deduced that the cluster of infections was chronologically and physically connected to the purchase and subsequent death of an imported parrot. Disease symptoms were consistent with *C. psittaci* infection. Epidemiological data supported the diagnoses and causes of death, despite the presenting symptoms sharing significant overlap with other common respiratory diseases.

There is growing awareness of the risks of epidemiological bridges in transmitting animal diseases to humans. Historical cases are a strong reminder of the fundamental role of scientific and public health responses in the face of such contagion.

The emergence of the SARS-CoV-2 virus and its subsequent global spread has placed renewed attention on the real risk that human–animal interaction poses to public health. While it is recognised that many animals, including agricultural and domestic species, can transmit infections to humans, the risk of pandemics arising from such interactions is generally low [1]. Nevertheless, the potential for global spread of some particular zoonoses has already been realised, such as in the case of the bacterial cause of plague, *Yersinia pestis*, in the thirteenth and later centuries [2]; and more recently the H5N1 avian influenza virus [3]. Zoonoses not only affect the health of individuals but have the potential to severely impact communities, socially and economically.

Psittacosis, a human respiratory disease transmitted to humans from infected birds, is perhaps a lesser-known example of a zoonotic disease. Psittacosis was first recognised in the 1930s when it was associated globally with numerous clusters of respiratory illness that led to high rates of human mortality and major disruptions to the global trade in parrots [1]. The disease, also known as pigeon-fancier's disease or parrot disease, is a human systemic infection caused by the intracellular bacterial pathogen, *Chlamydia psittaci* [4]. Today, infections in humans are typically the result of direct contact with the excretions of *C. psittaci*-infected parrots or domesticated poultry (chickens, ducks, turkeys) [5], although indirect contact with infected birds [6], as well as cross-host transmission from *C. psittaci*-infected mares and sick foals to humans have also been reported [7]. Although rare, there are also reports of human-to-human transmission following a putative initial avian-to-human transmission event [8–10].

Disease caused by this pathogen was first brought to widespread public attention in the period 1929–30 when the burgeoning global trade in parrots, prized for their colourful plumage for the garment and hat industry and as household pets, was found to

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cause what is now known as the 'Great Parrot Fever Pandemic' [1]. Countries that were impacted included the United Kingdom (UK), United States of America (USA), Argentina and Germany, with the source of the parrots traced back mainly to South America, but also Australia [1,11]. Globally, it is estimated that up to 800 people were infected during this pandemic, with around one hundred deaths [12]. Recognising the seriousness of the situation, many countries banned imports of parrots at the time unless for research [13]. Indeed, the potential lethality of psittacosis continued to be evidenced beyond the pandemic of 1929–30, by its naming as a disease useful in biological warfare by the United States War Department in 1947 [14].

The psittacosis pandemic in 1930 was well-recognised in port cities such as London, where imported birds were sold at local markets. Health reports for local London districts in 1930 included numbers of cases of psittacosis, alongside other infections of public health concern such as scarlet fever, smallpox, measles and influenza. We report here the epidemiological and clinical case details associated with one family group tragically impacted by the purchase of an infected bird in 1930, and discuss the wider public health impacts of psittacosis in a changing landscape where zoonoses are of increasing global concern.

1. Methods

Case details and autopsy reports were obtained from the National Health Service Trust archives at Barts Hospital in London. These include specific case notes recorded in the Director's Book of the London Hospital Pathological Institute, 1930. Other details were obtained from publicly available news media articles and 1930 publications of Medical Officer of Health Reports from London. The names of the affected individuals have been abbreviated as two-by-two nomenclature, i.e. the first two letters of their first and second names. While this is an attempt at some privacy, it is important to note that the full names of some family members were publicly reported and can be found in the public record.

2. Results

A timeline of events associated with this family cluster of psittacosis is presented in Fig. 1.

Ja Ro, aged twenty-seven years, from Barking, Essex, purchased a parrot from Spitalfields Markets in East London on 10 January 1930 and brought it home. The bird was in poor condition and remained unwell until its death eleven days later. The parrot was described by the mother of Ja Ro, namely El Ro, as, 'never had any life in it'. The case notes report that the parrot's 'feathers were very ruffled for three days previous to death'. Two other parrots purchased at the same time were taken to homes in Stepney and Romford. These parrots also died, with one being sent for laboratory investigations [15]. There are no details of cases of illness associated with these other birds.

The human cases attributed to psittacosis resided in three linked households. Ja Ro lived at home in Barking, Essex, with his mother El Ro (aged fifty-two years), sister Li Ro (aged thirty years) and brother, Ed Ro (aged eighteen years). A married sister, El Su, aged twenty-eight years, and in the third trimester of pregnancy, lived nearby in Barking, with her eighteen-month old son, and husband. A household help to the Ro family, H Ve, lived at Ilford, a town located about two km to the north of Barking.

The clinical features of the psittacosis cluster are presented in Table 1.

Within days following the death of the parrot, Li Ro and Ed Ro developed similar symptoms. Both individuals were subsequently admitted to hospital and later died. The 1930 report of the Medical Officer of Health for Barking describes the clinical features of illness of Li Ro thus:

'On January 25th, it was obvious that the girl was ill. There was a temperature of 101.6 and a pulse of 108; there was an intractable headache of the forehead, which did not radiate elsewhere; there was an intense fear of light; there was vomiting; the respirations were about 24; the heart showed no signs of disease other than those associated with the high temperature; the lung gave an ordinary note all over on being percussed, but with the stethoscope very fine signs of moisture could be heard over the lower parts of the lungs and the hila. The patient was on this day lying in bed in a semi-unconscious condition, from which it was difficult to arouse her. During the time the patient was in Barking the condition was progressive, the temperature became higher, the vomiting continued and the catarrh of the chest became more serious, so that on January 27th, the temperature was 103.8 and the pulse 120. The patient was clammy, hot and perspiring and the general conditions of the lung were those of a diffuse pneumonia.' [16].

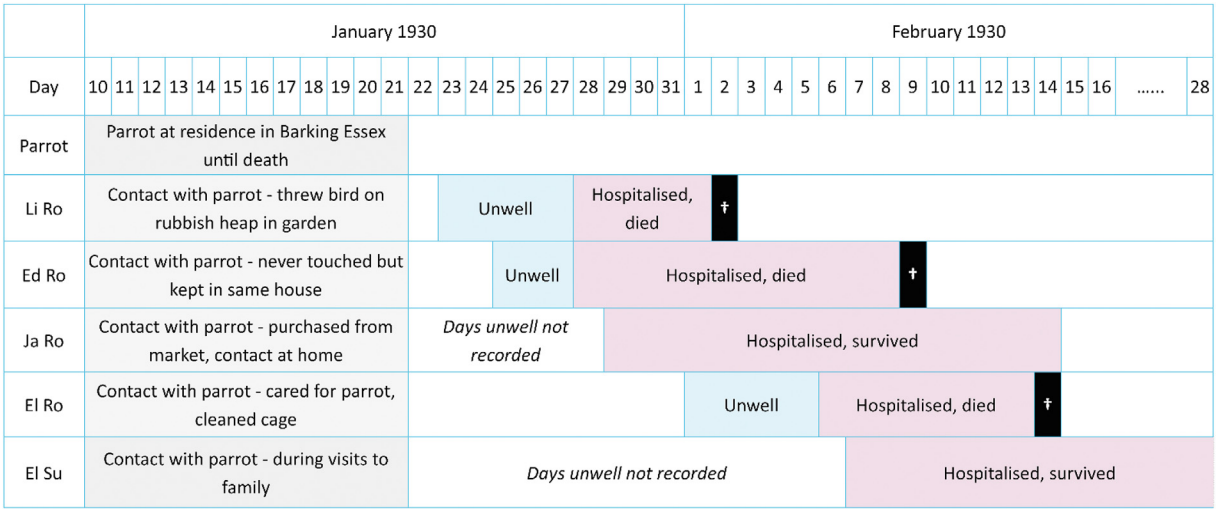

Fig. 1. Timeline of illness for psittacosis cases 1930. Note, a further case (H Ve), who worked in the Ro family house, was reported with three days illness, diagnosed as psittacosis during the same period.

Table 1
Clinical features reported in hospital notes for individuals diagnosed with psittacosis and associated with the Essex psittacosis cluster in January-February 1930.

Symptom	Li Ro	Ed Ro	El Ro
Fever (max)	106°F (41.1°C)	104°F (40°C)	103.5°F (39.7°C)
Headache	Yes	Yes	Yes
Photophobia	Yes	Yes	Yes
Cough	Yes	Yes	Yes
Muco-purulent sputum	No	Yes	Yes
Increased respiration	Yes	Yes	Not recorded
Lung sounds	Yes	Yes	Yes
Other	Sweating, nausea, vomiting, cyanosis	Collapse, severe epistaxis, increased pulse rate, bloody sputum, delirious, bleeding gums	Bronchitis, cyanosis, dyspnoea
Duration of symptoms prior to death	11 days	16 days	14 days
Cause of death	Heart failure. Haemorrhagic pneumonia. Septicaemia. Psittacosis.	Pneumonia. Septicaemia. Psittacosis.	Haemorrhagic pneumonia. Septicaemia. Psittacosis.

Surviving cases: Ja Ro was reported to have had a temperature of 102°F (38.9°C) and 17 days of illness; H Ve was reported to have had a temperature of 103°F (39.4°C) and three days of illness. No other symptoms were noted for these two cases. No case notes for El Su were located; however she was hospitalised for a lengthy period.

Ja Ro, who had purchased the bird, was admitted to hospital a day later than his siblings, remaining there for about two weeks until his discharge. Their mother, El Ro, developed similar symptoms to those of her children (Table 1), and was admitted to hospital where she later died. Few case details could be located for survivors El Su and H Ve; however, they, as well as Ja Ro, were diagnosed as psittacosis cases during February 1930. El Su was discharged from hospital on 28 February 1930 and delivered a healthy baby in the weeks following.

All patients reported a high fever with temperatures ranging between 102°F (38.9°C) and 106°F (41.1°C). Li Ro, Ed Ro and El Ro were all described as presenting with headache. The same individuals were also reported as having developed a cough with lung sounds noted (Table 1). Temperature, pulse and respiration data were available for two individuals, the brothers Ed Ro and Ja Ro, over the duration of their hospitalisation, and are compared in Fig. 2. Persistently high respiration, pulse and temperature were noted for Ed Ro who subsequently died, compared to his surviving brother Ja Ro.

Bacteriological tests were performed on specimens from Ed Ro during his illness. Agglutination reactions were reported in the Director's Book of the London Hospital Pathological Institute for 1930 as 'Negative to typhosus, Paratyph A + B, Aertrycke [Salmonella]. No Coli or Typhoid. No dysentery, No salmonella. No aertrycke.'

Post-mortem microbiological testing of various organ specimens was conducted to identify any aetiological agents associated with the three deaths (Table 2).

3. Discussion

The psittacosis cluster described in this report was one of similar outbreaks reported in the period 1929–30 on four continents, namely Europe (Germany, Switzerland, France, Denmark, Holland), Africa (Egypt, Algeria), North America (USA) and South America (Argentina) [1]. The mortality rate in the Essex cluster reviewed here was fifty percent. These results were similar to those reported from twenty cases of psittacosis documented in Hamburg in 1929, where the case fatality rate was thirty-five percent [17].

Patients with psittacosis typically develop symptoms within five to twenty-one days of exposure, usually ten days [18]. Based on analysis of eighty patients, the UK Ministry of Health report from

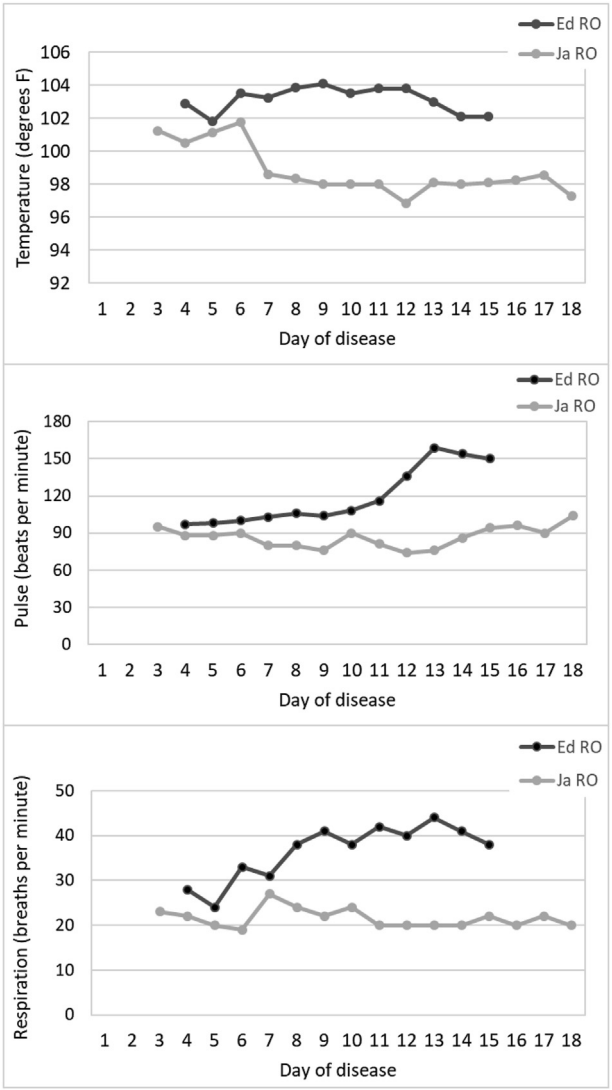

Fig. 2. Comparison of temperature, pulse and respiration data during hospitalisation for Ed Ro and Ja Ro.

Table 2
Bacteriological findings at autopsy for three cases associated with the Essex psittacosis cluster in January-February 1930.

Case	Bacteriological report at autopsy (verbatim transcript)
Li Ro	Pleural fluid grows *Staphylococcus aureus* and a short-chained streptococcus. Cultures from the lung grow *Staphylococcus aureus* and a short-chained streptococcus. Cultures from heart blood grow streptococci only. Cultures from liver and spleen grow a Gram negative lactose-fermenting bacillus. Jejunum no non-lactose fermenters present. All organisms are of the Coli group, mainly B. acidi lactici.
Ed Ro	Cultures from heart blood sterile. Cultures from liver grow a few *Staphylococcus aureus*. Cultures from lung grow numerous colonies of *Staphylococcus aureus*. Cultures from spleen grow a few colonies of *Staphylococcus aureus*.
El Ro	Cultures from heart blood remain sterile. Cultures from lung, liver and spleen grown a Gram-negative non-lactose fermenting bacillus which does not conform to the salmonella type. Upper jejunum. *B. proteus* and all the rest are lactose fermenting.

1930 regarded an incubation period of about 10 day as usual; however, the range could be from four to sixteen days [19]. In the Essex cluster, the first signs of clinical illness in Li Ro and Ed Ro were detected two weeks after the sick parrot arrived in their home, although the parrot was resident for some eleven days during that time and the incubation period could have been as short as two days. Indeed, Li Ro claimed never to have touched the bird except on the day it died. If initial contact with the parrot on 10 January 1930 was responsible for *C. psittaci* infection in Ja Ro and El Ro, the incubation period could have been up to twenty-five days. However, contact with contaminated feathers, droppings and dust at any time could have been responsible for infection. Also, there is good evidence to suggest that *C. psittaci* shedding in infected birds can be intermittent or persistent with excretion levels influenced by factors such as animal stress, placing further doubts over the exact time when the individuals in this cluster may have been infected [20]. Nonetheless, the apparent lengthy incubation period caused the Medical Officer for Health for Barking to suggest the disease could be ' … communicable not only from parrots to man but from man to man.' [16]. With growing evidence of the latter, this possibility cannot also be ruled out in this instance although disease was limited to the house cluster and no other cases (e.g. health workers) were reported [8–10].

The 1930 British Ministry of Health report describes an illness of about two or three weeks duration, but with pyrexia lasting as long as eight weeks in some cases [19]. Early symptoms of high fever, epistaxis, chills and generalised pain were said to be followed by exhaustion, a 'troublesome paroxysmal cough', 'crepitations and scattered rhonchi'. Persistent and severe headache were also frequently noted, as occurred in the Essex cluster. Mortality amongst the eighty cases analysed was about twenty percent [19] less than the fifty percent mortality of the Essex cluster. High fever, also noted in all individuals in the Essex cluster, was identified as a consistent clinical feature (seventy to one hundred percent) following a more recent review of 532 cases of psittacosis [21]. The comparison between the very poor vital signs of Ed Ro, who died, and better results for Ja Ro, who survived, demonstrates the serious symptomology and outcomes of the disease.

In the case of H Ve, the household help for the Ro family, the initial diagnosis was influenza until it became clear that she had been in contact with cases of psittacosis. While there was some consideration at the time that the disease was transmitted person-to-person in this cluster, it is highly likely that she attended the home during the time that the parrot was alive, or that feathers or droppings were present in the household on the days she worked. The broader confusion over the cause of this disease is likely to have contributed to a reasonable under-reporting of case numbers of psittacosis during this global pandemic. The Medical Health Officer of Barking noted the potential for cases in this cluster and elsewhere to be confused with cases of influenza given that:

' … psittacosis, as we have seen it, may be likened very closely to a case of influenzal pneumonia with cerebral complications, and it may be that we shall find that many cases of pneumonia of the influenzal variety are similar due to predisposing causes' [16].

In the Essex cluster, perhaps the most important aspect leading to the correct association to psittacosis, rather than influenza, was the local public health investigation that resulted in the identification of a sick parrot as a common factor. The identification of a vector is critical to understanding the epidemiology of zoonotic diseases and to developing successful strategies for controlling disease outbreaks. For psittacosis, a link to wild parrots continues to form part of the case definition of this zoonotic disease in Australia and elsewhere, with 'direct or indirect exposure to bird or bird products or contact with a confirmed human or animal case' considered to be epidemiological evidence of the disease [22].

While the 1930 outbreak was ongoing, there remained much speculation over the aetiological agent of the illness. A setback to an early identification of this pathogen in cases of psittacosis arose from a suggestion that the aetiological agent was similar to the cause of typhoid-like illnesses in humans. This notion arose from the work of Nocard, who, in 1893, cultivated Gram-negative bacteria rods from the dried wings of parrots that had died of suspected psittacosis during transport by sea [1,23]. The true aetiology of psittacosis would only be discovered later, in 1930, with the almost simultaneous and independent reporting by three different groups of investigators of the presence of small, filterable bodies consistent with a viral aetiological agent [23]. Detailed experimental studies in animals, conducted during the pandemic and in the subsequent five to ten years, would eventually lead to the description of infectious bacterial bodies that share a bi-phasic developmental cycle, and that we know today as belonging to the genus *Chlamydia* [24,25].

The classic scientific experiments and public health responses undertaken in response to a real health threat in the 1930s foreshadowed rapid advances in understanding and responding to major crises of contagion in subsequent years. For psittacosis, the combination of scientific studies and thorough public health investigation led to the identification of a novel pathogen, a common vector, and changes to legislation, administration and society. While the origin of SARS-CoV-2 remains unclear [26], the general consensus is that initial human infections are the result of 'spill-over' at some stage in the evolutionary history of this pathogen. Scientific investigation has led to the rapid identification of the causative agent of SARS-CoV-2 [27] and scientific study, again, has formed the basis of the ongoing public health investigation and response to the pandemic.

There are some notable differences between the recent SARS-CoV-2 pandemic, previous influenza pandemics and the 'Great Parrot Fever Pandemic'. First and foremost is the obvious difference in the scale of the outbreaks of these global events. As at October 2022, there have been over 617 million confirmed cases of SARS-CoV-2 spread across nearly all countries and all continents with 6.5 million deaths [28]. The globally distributed clusters of psittacosis reported in 1929–1930, on the other hand, are suspected to have only involved infection of 800 individuals, albeit with a significantly higher case mortality rate. While human-to-human transmission is the driver of the spread of SARS-CoV-2 and other viral respiratory pandemics across international borders, it is likely

that the globally distributed clusters of psittacosis were the result of discrete zoonotic transmission events with possible but likely limited potential human-to-human transmission. In this sense, while the outbreaks of 'parrot fever' are regularly referred to in scientific and popular press as a 'pandemic', this is probably a misnomer and the events during 1929–1930, while significant at the time, probably do not meet the modern definitions of a global pandemic.

Study of past pandemics is an important practice in preparedness for the future. For instance, the 1918 pandemic provided evidence of the impact of late lock-down on mortality and of the value of social distancing [29]; and the H1N1 2009 influenza pandemic identified risk factors for severe disease, such as obesity [30]. Effective surveillance was critical in smallpox eradication [31], and indeed the eradication program developed for smallpox has since proven successful for other diseases such as guinea worm [32]. In 2007, Heymann highlighted issues evident in the learnings from past pandemics, namely the "roles and responsibilities of health workers; the consequences of infectious disease to commerce among nations; the challenge of providing equitable access to health-care resources; and the balancing of individual rights versus public welfare" [33]. Pandemic influenza, Human Immunodeficiency Virus (HIV), Ebola virus, and the current SARS-CoV-2 pandemic exemplify the impacts of contagious diseases on various facets of everyday life, as well as on the health of specific populations. Responding to these threats has necessitated a range of specific actions, including the development of education tools to address social stigma of disease; and changes in healthcare and personal protection practices. Impacts of contagion also extend beyond the personal health level to include pressure on global economies reliant on trade and tourism for revenue [34]. Thus, in addition to epidemiological and scientific research and investigation, there is a need for study of past outbreaks to identify potential impacts at personal, societal, system and national levels when planning response strategies to mitigate future threats.

While different in scale to the viral pandemics of the last century, the Essex psittacosis cluster as but one of the many clusters of this zoonotic disease around the world in a relatively short period of time, further illustrates the impact of these diseases on the everyday life of individuals as well as on other levels of society. The precedents set and the successful outcomes achieved following investigations into historical zoonotic disease outbreaks are critical to our modern understanding of the global impact of infectious diseases and how public and population health strategies are needed to respond to the emergence of new infectious disease threats.

Declaration of competing interest

None declared.

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